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EXAMINER	
SITTON, JEHANNE SOUAYA	

ART UNIT	PAPER NUMBER
1634	

DATE MAILED: 09/26/2006

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/789,169	WEINBERGER ET AL.	
	Examiner	Art Unit	
	Jehanne S. Sitton	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☐ Responsive to communication(s) filed on *26 June 2006*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-51* is/are pending in the application.

4a) Of the above claim(s) *25-51* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-24* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date *9/2004*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Election/Restrictions

1. Applicant's election with traverse of Group I, claims 1-24 in the reply filed on 6/26/2006 is acknowledged. The traversal is on the ground(s) that the members of the group are sufficiently few in number that a search and examination can be made without serious burden on the examiner. This is not found persuasive because the methods elected are unrelated to any of the other groups presented. A search for group I is different from the search required for the other groups and is not coextensive. None of the other groups require the method steps of group I. As such, a search burden exists for searching each of the groups. As the different groups are patentably distinct for the reasons presented in the restriction requirement and a search burden exists for searching each of the groups, the requirement is still deemed proper and is therefore made FINAL.

Claim Rejections - 35 USC § 112

2. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

3. Claims 1-24 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention. There are many factors to be considered when determining whether there is sufficient evidence to support determination that a

disclosure does not satisfy the enablement requirements and whether any necessary experimentation is undue. These factors have been described by the court in *In re Wands*, 8 USPQ2d 1400 (CA FC 1988). *Wands* states at page 1404,

> "Factors to be considered in determining whether a disclosure would require undue experimentation have been summarized by the board in Ex parte Forman. They include (1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence or absence of working examples, (4) the nature of the invention, (5) the state of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims."

The nature of the invention and the breadth of the claims:

The claims (1 and 2) are drawn to a method of predicting the likelihood that any individual will have impaired or enhanced hippocampal function or verbal memory by obtaining a DNA sample and determining the presence or absence of a single nucleotide polymorphism (SNP) G to A resulting in the substitution of a methionine for a valine (G=Val, A=Met) at amino acid position 66 relative to the start of the precursor protein sequence in BDNF, wherein the presence of a G to A polymorphism resulting in a methionine residue at position 66 is correlated with impaired hippocampal function or verbal memory and wherein the presence of an A to G resulting in a valine residue at position 66 is correlated with enhanced hippocampal function or verbal memory. The claims (13 and 14) are also drawn to a method of predicting the likelihood that any individual will have impaired or enhanced hippocampal function or verbal memory by obtaining a biological sample containing the precursor BDNF protein or relevant portion thereof and determining the amino acid present at position 66 relative to the first amino acid of the precursor protein wherein the presence e of a methionine residue at position 66 is indicative of

impaired hippocampal function or verbal memory and wherein the presence of a valine residue at position 66 is indicative of enhanced hippocampal function or verbal memory.

The claims (claim 3) are also drawn to a method for predicting the likelihood that an individual has risk for or protection from schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory, obtaining a DNA sample and determining the presence or absence of a single nucleotide polymorphism (SNP) G to A resulting in the substitution of a methionine for a valine (G=Val, A=Met) at amino acid position 66 relative to the start of the precursor protein sequence in BDNF, wherein the presence of a G to A polymorphism resulting in a methionine residue at position 66 is correlated with risk for schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory, and wherein the presence of an A to G resulting in a valine residue at position 66 is correlated with protection from schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory. The claims (claim 15) are also drawn to a method of predicting the likelihood that any individual will have risk for or protection from schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory by obtaining a biological sample containing the precursor BDNF protein or relevant portion thereof and determining the amino acid present at position 66 relative to the first amino acid of the precursor protein wherein the presence of a methionine residue at position 66 is indicative of risk for schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory and wherein the presence of a valine residue at position 66 is indicative of protection from schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory.

The claims are further limited to individuals that are at risk for development of impaired hippocampal function, impaired verbal memory or at risk for development of schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory (claims 4-6, 16-18). The claims are also further limited to individuals that exhibit clinical symptomology associated with (claims 7-9, 19-21) or individuals that are clinically diagnosed as having (claims 10-12, 22-24) impaired hippocampal function, impaired verbal memory, or schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory.

The nature of the invention, therefore, requires the knowledge of a predictive association between the presence of an A/met or G/val at codon position 66 of BDNF and impaired or enhanced hippocampal function, impaired or enhanced verbal memory, or risk for or protection from schizophrenia, schizoaffective disorder, and any other psychotic and mental disorders involving impaired memory in any individual.

The amount of direction or guidance and presence/absence of working examples:

The specification teaches that BDNF is a neurotrophin and contains at least one known nonconservative SNP producing a met66val substitution (page 3). The specification teaches that verbal memory was assessed in 184 patients with schizophrenia, 283 siblings, and 101 controls and that NAA (N acetyl aspartate) was available for 110 subjects (page 4). The specification teaches that the effect of genotype was significant across all groups for memory scores ($p<.008$) and that the met allele was associated with poorer performance. However, the specification teaches that BDNF genotype had no effect on IQ or prefrontal cognitive measures.

The specification teaches that the met allele was associated with reduced hippocampal NAA ($p<.07$), however this result does not appear to be statistically significant. The specification further teaches that in two separate cohorts studied with fMRI, subjects with the met allele had abnormal patterns of hippocampal activation while performing memory tasks, compared to val/val homozygote subjects. The specification, however does not teach if this was statistically significant (page 4). Although the specification suggests that the met allele may be associated with impaired hippocampal function or impaired verbal memory, the specification does not provide any guidance or working examples that the val allele is associated with enhanced hippocampal function or verbal memory. The specification provides no working examples of human subjects and controls demonstrating that the val allele is associated with enhanced hippocampal function or verbal memory than in normal controls.

The claims are further drawn to determining the likelihood that an individual is at risk for or protection from schizophrenia as well as other disorders involving impaired memory. The specification teaches that the frequency of the deleterious met allele was slightly higher in patients with schizophrenia than controls ($p=.05$), however, this result is contradicted by the specification's teachings that in a TDT analysis, transmissions of met vs val did not differ significantly (page 4). Accordingly, it does not appear that the met allele is predictably indicative of disease risk or that the val allele is predictably indicative of disease protection, as broadly encompassed by the claims.

The specification teaches that disorders encompassed by the broadly claimed "psychotic and mental disorders involving impaired memory" include Alzheimer's disease, head injuries and normal aging (page 5, last sentence of para 0009). The specification, however, does not

provide any guidance or working examples that the met allele is indicative of risk for or that the val allele is indicative of protection from, any of these disease encompassed by the broadly claimed invention, including schizoaffective disorder.

The claims encompass analysis in any individual, which includes any human population, as well as any other species. The specification provides no working examples of association studies between the indicated alleles and different ethnic populations. Further, the specification provides no teaching or working examples of the presence of the indicated alleles in any other species or mammal such as mice, rat, dog, etc.

The state of the prior art and the predictability or unpredictability of the art:

Although the specification sets forth a number of substantially different hypothesis (page 9, para 0022) the specification does not teach the effect of val66met SNP on BDNF function. The association between the polymorphism and its effect on hippocampal function, verbal memory, and disorders involving impaired memory are unclear. The art at the time the invention was filed, does not make up for the deficiencies in the specification. Association of the met or val allele at codon 66 of BDNF with hippocampal function, verbal memory and disorders involving impaired memory, such as schizophrenia, or Alzheimer's disease (AD), in different populations is highly unpredictable as exemplified by the teachings in the art.

The post filing date art of Neves Pereira (Neves-Pereira et al; Molecular Psychiatry, vol. 10, pages 208-212, 2005) teaches that although the val66met polymorphism has been shown to alter gene function, the risk may depend on the haplotypic background on which the val/met variant is carried (see abstract). As evidenced by the haplotype analysis for SNPs in BDNF,

Sklar (Sklar et al; Molecular Psychiatry, vol. 7, pages 579-593, 2002) teaches that 8 BDNF SNPs, including the val66met (a39) polymorphism are found in 6 different haplotypes, which are found at different frequencies in different populations (see table 4). The study of Neves Pereira contradicts the teachings of the specification in that it teaches that the valine allele, as opposed to the methionine allele as disclosed in the instant application, is associated with schizophrenia in a Scottish population (see abstract). Likewise, Kent (Kent et al; Molecular Psychiatry, vol. 10, pages 939-943, 2005) teaches that the valine allele was found to be preferentially transmitted in ADHD (see abstract) and Ventriglia (Ventriglia et al; Molecular Psychiatry, 2002, vol. 7, pages 136-139) teaches that the homozygosity for the valine allele appears to confer an increased risk for AD (see page 137, first col).

Alternatively, Zhang (Zhang et al; American Journal of Medical Genetics, Part B, vol. 141B, pages 387-393, 2006) teaches that there was no association found for the val66met polymorphism and AD, affective disorders, or schizophrenia. Jonsson (Jonsson et al, Progress in Neuro-Psychopharmacology & Biological Psychiatry, vol. 30, pages 924-933, 2006) teaches that there were no significantly different allele, genotype, or haplotype frequencies between patients with schizophrenia and controls for the BDNF val66met SNP and teaches that further studies are needed to establish risk with schizophrenia (see abstract). Antilla (Antilla et al; J. Neural Transm, vol. 112, pages 885-890, 2005) teaches that a study by Egan et al, 2003 (applicants own work) showed that the BDNF G196A polymorphism was not associated with schizophrenia, but that a study by Hong et al 2003 reported that the val/val genotype was slightly more common in schizophrenia patients, at opposite with the teachings of the instant specification. Antilla teaches that in a study of Finnish patients, the G196A (val66met)

polymorphism was not associated with risk of schizophrenia, treatment response, or age of onset (see pages 888-889). Further, Antilla teaches that previous studies have suggested that the frequency of val66met polymorphism is different in different ethnic populations. In line with this teaching, the post filing date art of Bath (Bath & Lee, Cognitive, Affective, & Behavioral Neuroscience, vol. 6, pages 79-85, 2006) exemplifies the unpredictability of correlating hippocampal function, verbal memory, or disease risk in different populations, as is broadly encompassed by the claims. Bath teaches that cognitive and behavioral effects associated with the methionine allele have been shown to produce much more robust effects in Caucasians (page 81, col. 1, 2nd full para). Bath teaches that the methionine allele is not uniformly distributed across all ethnicities or all regions of the world and that Northern Europeans appear to be much more affected than Asian populations, despite the fact that a higher proportion of the Asian population carry this allele, suggesting that some ethnicities may compensate for the variation in the BDNF gene through some as yet unidentified mechanism and are thus less affected by the presence of the polymorphism (see para bridging pages 81-82). Further, the claims are broadly drawn to any individual which encompasses any mammal, however Bath also teaches that this polymorphism has not been found in any vertebrate species other than in humans. Accordingly, it appears that this mutation does not exist in other species.

The claims (13-24) are also drawn to assessing the precursor protein and determining the amino acid present at position 66, thus encompassing analysis of the BDNF protein (page 6, para 0013), using for example, antibodies that bind to one form of the gene product but not another (page 10, para 0025). However, neither the art nor the specification teach an antibody that is capable of specifically differentiating the BDNF valine66 from the BDNF methionine66 variant.

It is unpredictable whether the amino acid change would be sufficient to result in the production of antibodies that can differentiate between the two molecules. In some cases, an antibody elicited by one antigen can cross-react with a different antigen if the two different antigens share an identical or very similar epitope (Goldsby et al., 2003, p. 141). Thus, absent knowledge of the binding epitopes and the effects of the instant polymorphism on those epitopes, it is difficult to predict whether or not any generated antibody will be able to function to differentiate the two alleles in an assay. In the instant case, it is unpredictable as to whether or not an antibody would be able to differentiate between the two variants, a feature that is encompassed by the claimed invention.

The level of skill in the art:

The level of skill in the art is deemed to be high.

The quantity of experimentation necessary:

In order to practice the invention as broadly as it is claimed, one would first have to establish that a predictive relationship exists between the val66met polymorphism and impaired or enhanced hippocampal function or verbal memory or risk for or protection from schizophrenia, schizoaffective disorder, or any neuropsychiatric disorder associated with impaired memory or in *any* population. Given the conflicting results in the specification and the art as to a predictable association between the valine and methionine allele and different disorders or cognitive affects in different populations, such analysis would be replete with trial and error experimentation, the results of which are completely unpredictable. Additionally,

given the broad scope of the claims, the skilled artisan would also be required to screen for possible polymorphisms from other species to determine if such polymorphism exists and is predictably associated with the likelihood of disease, verbal memory, or hippocampal function as broadly encompassed by the claims. Given that the art teaches this allele has not been found in other species, as well as the teachings of the art that the affects of the allele appear to be influenced by the genetic background it is found in, the results of such analysis are completely unpredictable, as neither the specification, nor the art at the time of filing, teach how the allele is associated with the claimed phenotypes. It was not known whether the alleles themselves are functionally associated, or linked to some functionally associated alteration hundreds or thousands of nucleotides away. Additionally, given the teachings of the Goldsby, unpredictable trial and error experimentation would be required to practice the invention as broadly claimed with regard to protein analysis, additionally required for claims 13-24.

Therefore, in light of the breadth of the claims, the conflicting guidance in the specification, the high level of unpredictability in the art, the nature of the invention, and the quantity of unpredictable experimentation necessary to practice the claimed invention, it would require undue experimentation to make or use the invention as broadly claimed.

4. The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

5. Claims 1-12 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

The preambles of claims 1-3 recite "predicting the likelihood that an individual will have" impaired or enhanced hippocampal function, impaired or enhanced verbal memory, or risk for or protection from a number of different disorders, however the body of the claim recite that the alleles are "correlated with" the phenotypes found in the preamble. However, the term "correlated" encompasses both positive or negative correlation. Accordingly, it is unclear if the claims are drawn to predicting the likelihood that an individual will have a certain phenotype, or simply drawn to detection of the val or met allele at position 66 of the precursor protein for BDNF. It is suggested that the claims be amended to recite "indicative of" as set forth in claims 13-15.

Claim Rejections - 35 USC § 102

6. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (e) the invention was described in (1) an application for patent, published under section 122(b), by another filed in the United States before the invention by the applicant for patent or (2) a patent granted on an application for patent by another filed in the United States before the invention by the applicant for patent, except that an international application filed under the treaty defined in section 351(a) shall have the effects for purposes of this subsection of an application filed in the United States only if the international application designated the United States and was published under Article 21(2) of such treaty in the English language.

7. Claims 1-9 and 12 are rejected under 35 U.S.C. 102(e) as being anticipated by SklarII (Sklar et al; US Patent 6,458,541).

The claims are broadly interpreted as being drawn to detection of the val66met polymorphism in a subject. Given the broad recitation of "correlated with", the preamble of the claims has been given no patentable weight as it merely sets forth an intended use which is not set forth in the body of the claim itself. The claim only requires detection of a G to A polymorphism which results in a val66met polymorphism in the BDNF precursor protein, as well

as a broad correlation step. Sklar II teaches detecting an A/G SNP at position 424 in the sequence encoding the BDNF precursor protein in patients with bipolar disorder as well as neuropsychiatric disorders, such as schizophrenia, ADD (see col. 1, lines 30-35, lines 55-60, Figs 1A and 1B; para bridging cols 14 and 15). Sklar II teaches that this SNP encodes a val/met change in the encoded protein. It is noted that this position corresponds to the position recited in the instantly claimed invention. Sklar II teaches determining relative risk (col. 18, lines 54-59; correlating). With regard to claims 4-9 and 12, the patient population taught by Sklar II is inherently "at risk for" and exhibits "clinical symptomology associated with" the claimed phenotypes as bipolar disorder involves hippocampal function and verbal memory as well as impaired memory.

Claim Rejections - 35 USC § 103

8. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

9. This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

10. Claims 1, 4, 7, and 10 are rejected under 35 U.S.C. 103(a) as being unpatentable over Hariri (Hariri et al; Society for Neuroscience Abstract Viewer and Itinerary Planner, vol. 2002, 620.12, 8/19/2002) in view of Sklar II.

The 60/316,736 application does not provide for a method of predicting the likelihood that a subject will have impaired or enhanced hippocampal function. Hariri teaches detecting the met66val allele in BDNF in individuals and assessing hippocampal response. Hariri teaches that individuals homozygous for the val allele had a significantly greater response during a declarative memory task and that subjects with a met allele exhibited diminished response in comparison to subjects homozygous for the val allele. Although Hariri does not teach detecting the polymorphism in a DNA sample, Sklar II teaches detecting an A/G SNP at position 424 in the sequence encoding the BDNF precursor protein (see col. 1, lines 30-35, lines 55-60, Figs 1A and 1B; para bridging cols 14 and 15). Sklar II teaches that this SNP encodes a val/met change in the encoded protein. It is noted that this position corresponds to the position recited in the instantly claimed invention. Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to use the SNP genotyping method of Sklar II in the method of Hariri for the purpose of providing a reliable method of detecting the met/val polymorphism disclosed by Hariri.

Conclusion

11. No claims are allowed.

12. Any inquiry concerning this communication or earlier communications from the examiner should be directed to examiner Jehanne Sitton whose telephone number is (571) 272-

0752. The examiner can normally be reached Monday-Thursday from 8:00 AM to 5:00 PM and on alternate Fridays.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla, can be reached on (571) 272-0735. The fax phone number for this Group is (571) 273-8300.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

Patent applicants with problems or questions regarding electronic images that can be viewed in the Patent Application Information Retrieval system (PAIR) can now contact the USPTO's Patent Electronic Business Center (Patent EBC) for assistance. Representatives are available to answer your questions daily from 6 am to midnight (EST). The toll free number is (866) 217-9197. When calling please have your application serial or patent number, the type of document you are having an image problem with, the number of pages and the specific nature of the problem. The Patent Electronic Business Center will notify applicants of the resolution of the problem within 5-7 business days. Applicants can also check PAIR to confirm that the problem has been corrected. The USPTO's Patent Electronic Business Center is a complete service center supporting all patent business on the Internet. The USPTO's PAIR system provides Internet-based access to patent application status and history information. It also enables applicants to view the scanned images of their own application file folder(s) as well as general patent information available to the public.

For all other customer support, please call the USPTO Call Center (UCC) at 800-786-9199.

Jehanne Sitton
Primary Examiner
Art Unit 1634
9/5/06